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PHYSODERMA DISEASE OF CORN

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INTRODUCTION

In recent years the Physoderma disease of corn (*Zea mays*) has been reported as doing considerable damage in the southern part of the United States.[1] The uncertainty as to the distribution of the disease and its economic importance, together with the lack of a knowledge of the life cycle and parasitism of the causal organism and the possibility of its becoming a serious pest in the Corn Belt, led the Office of Cereal Investigations to undertake an exhaustive investigational study of the problem. This work was undertaken by the writer in December, 1916. Since that time certain phases of the problem have been more or less completely developed, while others are in need of further study.

HISTORY OF THE DISEASE

Shaw (*8*)[1] in 1912 reported the occurrence of the disease in India as early as 1910, and gave a short description of the causal organism. At the annual meeting of the American Phytopathological Society at Cleveland, Ohio, 1912, Barrett[2] reported the occurrence of the disease in Illinois in 1911. In a personal interview Barrett stated that he received specimens of diseased corn from Ohio and North Carolina. Barre (*1, p. 23*) states that the disease was known to be present in South Carolina as early as 1911, and since that time has been doing considerable damage. Reports of the occurrence of the disease in Georgia in 1910 have come to the writer indirectly, but he has never been able to confirm them. There is no reason, however, to doubt its occurrence in Georgia at that time, since it is now known to be so widespread throughout the country. Prof. J. M. Beal, of the Mississippi Agricultural College, noted the disease in Mississippi as early as 1914. Mr. A. P. Spencer, of the Florida Agricultural College, claimed that considerable damage was caused by it in Lake County, Florida, in 1915. In the summer of 1915 Melchers (*6*) collected

[1] Reference is made by number (italic) to "Literature Cited," p. 141.

[2] BARRETT, J. T. PHYSODERMA ZEAE-MAYDIS SHAW IN ILLINOIS. Not published. Reference to title only in Phytopathology, v. 3, no. 1, p. 52. 1913.

specimens of the disease at Manhattan, Kansas, but, not being sure of its identity, kept the specimens until 1917 before making his report. A number of farmers throughout the South have told the writer that the disease has been present on their farms for many years. It was no doubt present in this country a long time before being reported by pathologists.

DISTRIBUTION AND PREVALENCE

In the third issue of the Plant Disease Survey Bulletin (*9, p. 52*), September 15, 1917, the writer published a map showing the known distribution of *Physoderma zeae-maydis* and the localities in which there was noticeable damage. The disease at that time seemed to be rather thoroughly

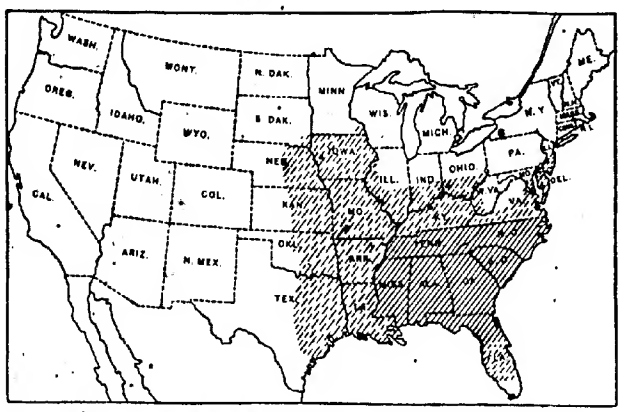

FIG. 1.—Map showing the distribution of *Physoderma zeae-maydis* in the United States. Broken lines, *P. zeae-maydis* present; solid line, *P. zeae-maydis* causing damage.

distributed throughout the Southern States as far north as North Carolina and Tennessee and west to the Mississippi River. Only single instances of its occurrence were known in Illinois and Ohio. Melchers (*6*) since that time has reported its occurrence in Kansas as early as 1915. In September and October, 1917, in cooperation with the Office of Plant Disease Survey, a detailed study was made of the prevalence and importance of the disease in representative localities of the infested area of the Southeast, not including Florida. The survey was also extended to determine the distribution of the disease throughout the United States. The disease was found to be prevalent practically throughout all localities where the detailed survey was made, very few fields being entirely free from it. The writer found diseased corn plants on the Blue Ridge Mountains of North Carolina at an elevation of about 3,000 feet. However, it was much less abundant than on the lowlands of the State. In the more extensive survey the disease was found to occur in Arkansas,

Delaware, Indiana, Kentucky, Maryland, Minnesota, Missouri, Nebraska, New Jersey, Oklahoma, South Dakota, Texas, Virginia, and West Virginia. It was also found in other sections of Kansas, southern Illinois, and Ohio. The disease, however, has not yet been found beyond the eastern part of Texas and Oklahoma, northward to southeastern South Dakota and Minnesota. Likewise the northern border of the infested zone extends from southern South Dakota and Minnesota through southern Illinois, Indiana, Ohio, West Virginia, and Virginia, and along the coast regions of Maryland, Delaware, and New Jersey (fig. 1). The disease is apparently much less prevalent in the area west of Mississippi, and north of North Carolina and Tennessee. The nature of the survey, being less intensive, may be responsible to a certain extent for this conclusion. It is possible that the disease has spread almost, if not quite, to its northern and western limits as permitted by certain weather factors.

ECONOMIC IMPORTANCE

The nature of the fungus causing the disease is such that its ability to produce serious injury to corn is limited by weather conditions, and in any ordinary season only local damage need be expected. However, in certain humid sections of the South, where the time for corn planting may extend over a period of three or four months in each year, weather conditions will more than likely be such as to favor a serious development of the disease on some of the plantings. This holds true especially for the South Atlantic and Gulf coasts and lower Mississippi Valley.

Barre (*2, p. 25*) says:

> The corn disease caused by *Physoderma* sp. as mentioned in the report last year has caused serious loss again this year. This disease was collected during the past season at a number of widely separated points in the state and seems to be more wide spread than ever before. This disease certainly deserves some attention and it is hoped that an investigation of its life history and habits can be undertaken in the near future.

The disease was known to be causing loss to the corn crop in Florida and Mississippi in 1915.

During the survey of 1917 the most pronounced losses were found along the Atlantic and Gulf coasts and in the Mississippi Valley. In the lowlands of North and South Carolina, and in the Gulf and Delta sections of Mississippi, frequent reports of as much as 5 per cent loss were given by the survey men. In some cases the damage was estimated at 6 to 10 per cent of the crop. The writer visited a few fields in the eastern part of South Carolina where the damage was perhaps as much as 10 per cent. Fields of this kind, however, were seldom found. These estimates were based entirely on grain loss, whereas the foliage, which is not considered of great importance except where the plants are used for silage, etc., was often badly injured. Smaller areas sustaining con-

siderable damage were reported throughout the Southeast, and as far north as the Mississippi Valley sections of southern Illinois and Missouri. These areas were usually very limited and were confined for the most part to low, wet lands. Considering the infested area as a whole, however, the percentage of damage, at least in the year 1917, was not very great.

In 1918, up to July 15, the disease had not developed to any great extent in the South. This fact was due, no doubt, to the very dry season in that section, which will be discussed later. However, mid-July is not too late for considerable injury to develop should conditions so change as to favor the disease.

FACTORS INFLUENCING THE DEVELOPMENT OF THE DISEASE

Seemingly the more important factors in the development of the disease are moisture and temperature. The fungus requires considerable moisture, with a fairly high temperature, for a high percentage of germination and infection. If these conditions are realized before the corn plants are more than half-grown, the disease probably will become severe if there is abundant spore material present. The following information regarding these factors has been noted:

(1) Serious injury has been confined largely to the South, where the summer temperature is continuously high, and to localities in which there has been considerable rainfall during the early growth of the corn crop. Plants may become infected in the later stages of growth, but the damage is not likely to be great in cases of this kind. The warm summer showers which may occur daily for a week or more furnish ideal conditions for the development of severe attacks by *P. zeae-maydis*. For instance, in the coast district of the Carolinas, where the disease was most severe in 1917, there was considerable rainfall in early summer. This would also hold true for the Delta in Mississippi and for southwestern Tennessee, where there was considerable rainfall in early June.

(2) Where the seasons were dry the disease was more pronounced on corn growing near water, or on low, wet land where the atmosphere was moist. Plants growing under these conditions are more likely to retain the sheath and bud water until the spores can germinate and produce infection. Conditions of this kind were noticeable at the South Carolina Station in 1918, where corn on the low bottom lands had considerably more infection than highland corn. In the lowlands the foliage of plants is less subject to drying by winds.

(3) Where the early corn season was dry and the late season wet the disease was more severe on late corn, and the reverse. Striking examples of the former were noted at the Mississippi Station in 1917, where there was less than 10 per cent infection on early corn and as high as 40 per cent infection on late corn, and at the Kansas Station, where early corn was almost free from the disease, while late corn showed considerable

infection. The latter, however, might have been influenced by temperatures. Early corn at the Pee Dee Station at Florence, S. C., was dwarfed by dry weather in 1918 and was not attacked to any great extent by the fungus. At Clemson College, S. C., very early and very late corn sustained considerable injury in 1917, while corn of intermediate ages suffered much less damage. The midsummer was very dry at this station, while the early and late seasons were rather wet.

(4) Apparently the more vigorous plants in certain cases sustain the severest attacks. These plants, however, will not continue to look vigorous after the disease has had time to develop. The fact that these vigorous plants are capable of shielding the free water which is held behind the sheath and around the growing point, or bud, from the drying effects of the wind and sun offers more favorable conditions for spore germination and no doubt accounts for the greater percentage of infection on plants of this type. In low, wet fields small plants are injured the same as large ones. This greater injury to large, vigorous plants was more noticeable in dry territory.

(5) Where seasons were wet—for instance, in the sections where greater damage was done in 1917—there was little noticeable difference in the amount of infection on corn growing on high and on low lands. At the South Carolina Station the most severe injury was caused to very early corn grown on comparatively high land. As previously mentioned, the early season was fairly wet at this point.

(6) The disease was found on the Blue Ridge Mountains of North Carolina at an elevation of about 3,000 feet, where it is claimed that the summer nights are always cool. In 1917, corn foliage was killed by frost on September 11 at this point. Very little of the disease was found at this elevation, however, even on wet lands. The disease is probably held in check to a certain extent by low temperatures which prevail at that elevation. A similar explanation was offered by the writer, in a summary of the survey work which was given by Lyman (*4*, *5*), for the absence of serious injury by the disease in the Northern States. It was also thought probable at that time that the disease had reached its northern limits. This supposition was drawn from the results of temperature studies of the germination of sporangia in the laboratory. Since that time, however, further investigation has shown that the sporangia of the fungus will germinate at a considerably lower temperature than was then supposed. However, the minimum temperature at which they are known to germinate is rather high (23° C.) as will be explained later, and it is probable that this temperature does not occur commonly during and immediately after the cold rains of early summer in the north. So far as is known at present, it would require a temperature not lower than 23° C. continuously for three days, with sufficient surface water for germination, in order for severe attacks to develop provided the sporangia are present on the plants. There is a question

as to whether these conditions are realized to any great extent in the Northern States, and it is hoped that the disease will not become a serious one in the Corn Belt.

(7) The rare occurrence or absence of the disease farther west is no doubt due to the semiarid conditions which exist there. The moisture requirements suitable for the development of an epidemic of the disease perhaps are seldom, if ever, realized in this section. However, further investigations are needed to determine in detail what the weather conditions are for the given sections and to study the possibilities for further development and spread of the disease in the Corn Belt.

HOSTS

So far as is known, all varieties of corn, including pop corn and sweet corn, are susceptible to the disease. Of the numerous varieties observed in the South there seems to be little, if any, difference in the degree of susceptibility shown by them. *P. zeae-maydis* also occurs on teosinte (*Euchlaena mexicana*), a near relative of the corn plant. It is possible that the disease was introduced from Mexico or Central America with this plant. The fact that it has been found in considerable quantities on corn in comparatively isolated fields where corn was never grown before and where no corn products were applied to the land suggests the possibility that there are other hosts for the fungus among wild plants.

SIGNS OF THE DISEASE

The disease occurs on the blade (Pl. A), sheath, and culm (Pl. B), and in rare cases it has been seen on the outer husks of the ears. Infection is usually more abundant on the lower half of the plant. Its first appearance on the thin parts of the blades resembles the early stages of the corn rust caused by *Puccinia sorghi*. It is first evidenced by slightly bleached or yellowish spots, which become darker within a few days when sporangia are formed. This darkening continues until the spots are brown to reddish brown, with a somewhat lighter margin. These spots are very small, seldom becoming more than 1 mm. in diameter, except where two or more of them coalesce. The spots may in some instances be so numerous as to give the entire blade a rusty appearance. For this reason the disease is often considered a true rust by persons who are not familiar with its nature. This rusty appearance is not uncommonly seen in bands across the blades, owing to the nature of infection, which takes place through zoospores in the bud water. On the midrib of the blade and on the sheath the spots become considerably larger. Often a single spot will measure 0.5 cm. across. They are irregular in shape and sometimes may be almost square in outline. This is due to the fact that they are definitely limited by the cell walls. In the very early stages these spots are evidenced by a color which is a somewhat darker green than the normal tissue surrounding them. This seems to indicate a

stimulating effect caused by the presence of the invading fungus. A few days later these spots are dark brown in the center, owing to the formation of the dark brown sporangia of the fungus. This change in color spreads until the entire spot is a dark or chocolate brown. These infections are often so abundant as to coalesce, and sometimes the entire sheath may become brown (Pl. 10). Where the infections are as numerous as this, the entire leaf often is killed before the plant is mature. However, the disease is usually more abundant on the parts of the sheath which are beneath the overlapping parts where the moisture is held. The disease often is accompanied by a reddening of the sheath and midrib, and especially the latter, which may almost entirely mask the brown spots. After the plants begin to mature, the epidermis becomes loose over these areas and they appear as brown blisters. This dry epidermis breaks easily and the spores are liberated as a brown spore dust. The entire parenchyma tissues of the invaded parts are destroyed by the disease, leaving the vascular system as so many free threads after the spores have been liberated (Pl. 11, C, E). On the culms the spots are very much like those on the sheath and midrib. They are usually more abundant at the nodes and just below the nodes, where spores are more likely to lodge and where free water is held by the sheath. The culms often are completely girdled at the nodes and are very easily broken by the winds after the tissues have been invaded. The disease is responsible for considerable lodging of corn in the South in the early stages of maturity. Only the lower nodes as a usual thing become so thoroughly invaded by the fungus as to be easily broken (Pl. 11, A, B). Considerable damage may result from severe attacks of this kind. After the plants have fallen, the pith at the infected nodes will be found to be filled with a brown mass of spore material (Pl. 11, D).

The signs of the disease on teosinte (*Euchlaena mexicana*) are very similar to those on corn, and therefore a separate description will not be necessary.

The pronounced signs of the disease have led farmers to apply various significant terms to it in the way of common names. The writer has heard the following names applied to it: "Rust," "corn measles," "corn pox," "dropsy," "frenching," and "spot disease." None of these terms, however, is in general use, and some of them—for instance, "rust" and "frenching"—would be incorrect, as corn is known to be affected by other distinct diseases called by these names. The name "falserust" has been suggested as a desirable common name for the disease. There would be a strong tendency, however, on the part of the layman to drop the word "false," thus causing a confusion with the true rust. Furthermore, the disease on the sheath and the culm bears very little resemblance to a rust. Since no satisfactory term suggests itself at present and since the scientific term "Physoderma," seems to be gaining favor as a common name, the author suggests that this term be retained.

CAUSAL ORGANISM

There still remains some doubt as to the correctness of the classification of the causal organism. According to the description of Cladochytrium and Physoderma as given by the leading mycologists, the organism evidently belongs in one of these genera. The essential difference between the two genera lies in their method of reproduction. The genus Cladochytrium may have both thick-walled sporangia, or so-called resting spores, and thin-walled sporangia, or presporangia, while the genus Physoderma is characterized by having only thick-walled sporangia (resting spores). As the species on corn is not known to produce the thin-walled sporangia, its thick-walled sporangia definitely place it in the genus Physoderma. Shaw's (*8*) description of the species from India was based almost entirely on the resting spores (sporangia). Barrett [1] found the disease in the State of Illinois in 1911 and in 1912 declared the organism identical with the species described by Shaw. Measurements of the sporangia of the fungus in America are practically identical with those given by Shaw (*8*). Measurements given by Shaw are 18 to 24 by 20 to 27 μ, while the writer finds the sporangia of the organism in this country to measure 18 to 24 by 20 to 30 μ. Therefore, so far as size of sporangia are concerned, the fungus in America is apparently identical with that described by Shaw from India as *Physoderma zeae-maydis*.

DESCRIPTION OF THE ORGANISM

The sporangia of the fungus are smooth, brown, thick-walled, 18 to 24 by 20 to 30 μ, slightly flattened on one side where the outline of a definite cap or lid can be seen by careful observation. On germination this trapdoor lid opens, or is carried up by the top of the thin-walled endosporangium, which finally ruptures at the apex and liberates a number of uniciliate zoospores. These zoospores are 3 to 4 by 5 to 7 μ, with a cilium three to four times the length of the spore itself. The zoospores have a comparatively large central oil droplet or nucleus. After their active stage these motile spores come to rest, in most cases lose their cilia, spread slightly in an ameboid fashion, and germinate by putting out fine fibrous hyphae. The mycelium is composed of very fine fibers, about 1 μ thick, which connect the large vegetative cells which Clinton (*3*) and Von Minden (*7, p. 397–410*) term "*Sammelzellen*." These enlarged cells, which may occur singly or in groups of two or more, produce sporangia directly or send out short fibers which produce terminal sporangia. The fungus is apparently an obligate parasite, and the mycelial stage is seen only within the tissues of the host plant. After the sporangia are mature no traces of the mycelium can be seen (Pl. 17, *B*).

[1] BARRETT, J. T. *op. cit.*

GERMINATION OF THE SPORANGIA

The more essential factors influencing sporangium germination and zoospore formation are moisture, temperature, and fresh air. After the sporangia have become thoroughly dried, it is very difficult to obtain germination. Sporangia which have just matured germinate readily when taken directly from the green corn plant. Germination was best obtained by placing the sporangia in a small amount of water in a watch glass or other shallow vessel, which was in turn placed in a large moist chamber. The sporangia seemed to germinate equally well in either distilled or tap water. The moist chamber was kept in an incubator or placed in the open room where the temperature was high enough for the germination of the sporangia. An incubator, in order to give good results, should be large and well regulated so as to keep the air as fresh as possible. The moist chamber should be kept thoroughly damp and should be rather large. The temperature should be kept constantly between 23° and 30° C., and preferably at 28° to 29°, as this seems to be the optimum range of temperature for germination. Sporangia placed as nearly as possible under these conditions often fail to germinate for some unknown reason. At other times sporangia from the same source germinate readily.

With the proper conditions of temperature, moisture, etc., the endosporangium absorbs water and begins a process of swelling, which causes the lid or cap of the resting spores to open in doorlike fashion or to be carried at the apex of the protruding endosporangium (Pl. 13, *b–d*). The lids begin opening in from 30 to 48 hours after the sporangia have been placed in the incubator. The granular content of this endosporangium begins rounding up into small nuclei, or oil droplets, which finally become the central bodies of the zoospores. Within a few hours after the sporangia start opening, zoospores are formed with the small oil droplets as centers. A small projection, or papilla, develops at the apex of the thin-walled endosporangium, and after the zoospore formation is complete a movement of the contents of the endosporangium can be seen to take place toward the projection, which breaks open, whereupon the zoospores are liberated in rapid succession (Pl. 13, *c*). These zoospores, which are usually 20 to 50 in number, swim away at first with a very jerky motion, which gradually becomes more uniform until their motile stage has ended. This occupies from one to two hours, depending somewhat on the temperature to which they are subjected. With cooler temperatures their active period is shorter. After their motile stage is over they settle down and in most cases lose their cilia and spread slightly in an ameboid fashion before germinating. The various stages of germination and zoospore formation and activity were studied with the aid of the high power lens of the microscope which was immersed in the zoospore suspension contained in a watch glass. The water in this case serves very nicely as an

immersion fluid. After the majority of the zoospores have escaped from the zoosporangium, it is very often the case that the collapsing wall of the endosporangium catches one or more of them within, where their motile stage can be easily studied (Pl. 13, *f*). A single zoospore has plenty of room to swim around within the empty sporangium.

ZOOSPORE GERMINATION AND HOST PENETRATION

Within one to two days after the zoospores have come to rest, they begin to germinate by sending out very fine, fibrous hyphæ (Pl. 13, *h*), which cease to grow after they have reached a few microns in length if they fail to come in contact with the host plant. Few of them ever reach this stage if they are kept in ordinary tap water, for they serve as a prey to bacteria and numerous protozoa. They break down completely and apparently disappear under adverse conditions. The small hyphæ which were produced by the germinating spores were made visible by applying to the spore solution a few drops of 5 per cent potassium iodid with enough iodin dissolved in it to give the solution a dark-brown color. This gradually killed the zoospores and stained them slightly at the same time. This method was used for staining both the cilia and hyphæ of the zoospores, which are so small that it requires a high power lens to make them visible. In this case the immersion lens was used in the manner previously described.

If the zoospores are in contact with the host epidermis, the fibers continue to grow after germination and penetrate the epidermal cell walls, thereby producing infection (Pl. 14). Very delicate technic was required in order to determine this point. The bud of a young corn plant was unfolded until the very thin white tissue, which was free from cholorphyl, was obtained. Sections somewhat smaller than a cover slip were cut from this thin leaf tissue and placed on slides. Drops of a zoospore suspension containing numerous spores were placed on these thin sections. The slides were then placed in the moist chamber and incubated at the same temperature required for resting spore germination. After two days a drop of the iodin solution was placed in the drop of spore suspension, a cover slip placed over the section, and an examination made. It was necessary to use the immersion lens to see what was taking place. Oil could not be used on a loose cover placed in this manner because it was so viscous as to hold the cover slip while the slide was being moved, and thereby disturbed the sections. A drop of water was used instead of oil and was found to be fairly satisfactory. At the end of two days the fine, fibrous hyphæ had, in cases, penetrated the epidermis, and some had produced the large swollen cells within the epidermal cells of the host (Pl. 14, *c*, *e*, *f*). The germinating zoospores were more commonly found attached to the host near the dividing wall of two epidermal cells. In some instances more than one hypha was seen passing through the host cell wall from a single zoospore (Pl. 14, *a*, *b*, *d*).

DEVELOPMENT OF THE FUNGUS WITHIN THE HOST TISSUE

So far as is known, the fungus is an obligate parasite. After zoospore germination and host penetration the fine mycelial fibers invade a number of the surrounding parenchyma cells, forming numerous enlarged cells, or *Sammelzellen* (Pl. 15). These enlarged cells are always intracellular, and are often in groups of two or more. A number of the small fibers are usually found extending in various directions from these cells. These fibers penetrate the host cell walls at any point (Pl. 16, *a–e*), passing directly into the adjoining cells which are likewise invaded through the same process of enlarged cell and fiber formation. Commonly where the host cell walls are penetrated the cell wall and the hypha of the fungus seem to be unmodified. In some cases, however, there may be a slight enlargement of the mycelium or a slight thickening or modification of the host cell wall, or perhaps both (Pl. 16, *a–e*). The hyphæ of the fungus are so small that no opening can be seen where they pass through the cell walls. The enlarged cells of the fungus may apparently develop directly into sporangia or send out short fibers which produce a single terminal sporangium. In cases where the enlarged cell develops directly into sporangia there is a rounding up of the content of the enlarged cells around a denser part of the protoplasm, which is to all appearances a nucleus. Where fibers grow out to produce terminal sporangia, they may arise directly from the enlarged cells without any noticeable disturbance to the nuclear structure. In some instances a double nucleate condition is seen, and a thread or fiber develops from this structure to produce a terminal sporangium (Pl. 16, *g*). A very common form of the enlarged body is an elongated structure containing from two to four cells (Pl. 16, *f*, *g*), but more commonly two cells. These structures, which have very thin walls and less dense protoplasm than some of the more compact structures, are often found to produce sporangia on the thin fibers. Where there are only two cells present one may give up its contents and collapse while the other may develop directly into a sporangium or produce a sporangium at the end of a hypha. In some cases both cells reproduce in one way or the other. The same is true for the three-celled and four-celled bodies, one or two cells of which apparently may collapse while the others produce sporangia. The double nucleate condition was more noticeable in structures of this kind.

After the formation of sporangia is complete, the invaded host cells are usually filled with them (Pl. 17, B). No traces of the mycelium or vegetative cells can be seen at this stage. These parts are entirely absorbed or broken down in the process of reproduction. The host cells appear to be slightly enlarged, owing to invasion by the fungus. They die as soon as the sporangia are formed, as their protoplasm is almost completely destroyed by this time. In the early stages of invasion there is a stimulation of the invaded cells which is brought about by the presence of the parasite. This no doubt accounts for the noticeably slight enlargement of cells.

FIXING AND STAINING METHODS

Material was obtained from the different parts of infected plants at various stages of the development of the disease and killed, preferably in Flemming's medium fluid. The material was put through the regular process of washing until it had been passed through 70 per cent alcohol. It was then placed in a 10 per cent solution of hydrofluoric acid and allowed to remain for three or four days to remove any silicates which might be present. Sections not treated in this manner injured the knife and could not be sectioned satisfactorily. The material was then returned to 70 per cent alcohol and the regular process followed until it was embedded in paraffin.

Three different stains were used for differential staining of host and fungus tissue—namely, Delafield's hematoxylin, and Flemming's triple and Pianeze stains. Both the triple and Pianeze stains proved to be desirable for this purpose, while the hematoxylin was not satisfactory. The orange G in the triple stain was taken up very readily by the fungus and was very desirable for staining the small hyphæ. The Pianeze stain gave the fungus tissue a pinkish color and served to bring out more of the details of structure, especially in the reproductive bodies. Sections from both corn and teosinte were stained, and the more successful sections were obtained from the thicker parts of the sheath tissue of teosinte.

ARTIFICIAL INOCULATIONS

Artificial inoculations were first tried in the greenhouse at Madison, Wis., in the winter of 1916–17. A special section of the house was obtained for this work, one in which a fine spray of water was kept going to keep the room damp. The temperature was kept at as near 30° C. as possible, which was perhaps a little too high for the germination of the sporangia. The plants were inoculated by spraying a suspension of sporangia behind the sheaths and in the bud. None of these plants were infected, for some reason. Inoculations were made in a similar manner on plants in a small isolated plot at West Raleigh, N. C., on July 23, 1917. At the same time these inoculations were made there was a daily occurrence of summer showers which continued three days after the date of inoculation. These plants were kept under observation by Dr. F. A. Wolf, Pathologist of the Station, who observed the first signs of the disease 10 days after the inoculation had been made. Two weeks after the inoculations had been made, he reported the presence of the dark-brown spots caused by the presence of abundant sporangia. The writer received specimens from these plants for his collections. In May of this year (1918) inoculations were made on corn growing in the greenhouse at the Arlington Experimental Farm at Arlington, Va. The weather was very warm, and it was necessary to spray these plants twice daily in order to keep the bud and sheath water from evaporating. On the writer's return

from a field trip four weeks later, June 14, he found that the disease had developed to a more or less extent on most of the plants which were inoculated (Pl. 12).

In cases where infections were obtained, the upper leaves, which were in the bud at the time of inoculation, showed the disease near their tips, while leaves which were mature were diseased at the base. This explains in part the fact that in the field the disease may be present only on the tips of blades or confined to the basal parts. The occurrence of the disease in bands of alternating heavy and light infection across the blades, which is often very noticeable, is no doubt due to the effect on sporangia germination of alternating periods of favorable and unfavorable temperature and moisture conditions while the leaves are emerging from the bud. The part of the blade which happened to be in contact with the bud water while the zoospores were being liberated became infected.

OVERWINTERING OF THE SPORANGIA

The sporangia of *Physoderma zeae-maydis* pass the winter in the old infected plants and in the soil, and germinate the following summer to produce new infections. Through the kindness of Dr. H. E. Stevens, of the Florida Agricultural College, fresh sporangial material was obtained from Florida at four-week intervals from January to April, 1917. These sporangia were found to be viable each time. Sporangia collected at the Alabama and Mississippi stations in May and June of the same year germinated readily. Sporangia buried about 3 inches deep at Clemson College, S. C., September 7, 1917, germinated in small percentages as late as July 20, 1918. These sporangia were taken from the soil on July 10. Sporangia left aboveground at the same point germinated also. Sporangia taken from the field on January 5 at Agricultural College, Miss., and buried 3 to 5 inches deep germinated about 50 per cent on June 20. Sporangia which remained aboveground in an open box gave a lower percentage of germination. This may be due to the fact that the sporangia aboveground were much drier when the tests were made. The winter was severe at this point, the temperature at one time being as low as zero Fahrenheit. This temperature apparently does not injure the sporangia, as the author had allowed sporangia to freeze in a cake of ice at the Wisconsin Station when the temperature was $-8°$ F. After several days the ice was melted from the sporangia, and some of them germinated. Sporangia collected in Alabama, Florida, and Mississippi in June, 1918, showed a high percentage of viability. Sporangia collected in South Carolina as late as July were found to be viable. Later tests than these have not been made, and it may be possible that these sporangia live in the old infected plants and in the soil for a number of years. Material has been prepared for further tests.

DISSEMINATION

This fungus, as is the case with many others, is no doubt disseminated in numerous ways. After the infected plants are mature the sporangia are liberated in large quantities and are free to be carried by such agencies as wind, running water, insects, and various animals, including man.

Wind is certainly responsible for considerable dissemination of sporangia. This was demonstrated, as will be shown by Tables I and II. Table I shows the results of sporangium catches on common microscopic slides at Agricultural College, Miss., in January, 1918. These slides were coated on one side with ordinary vaseline (a method used by Mr. H. D. Barker, of this Office, for catching wind-blown spores) and were placed on stakes 1 inch square at heights of 1, 2, and 3 feet. A slide was placed on each of the four sides of the stake at the different heights and held in place by rubber bands passing around each end of the four slides and the stake. The stakes were placed in the field so that the slides were facing the four points of the compass. A larger number of spores were caught on the slides facing the prevailing winds at the time the experiment was conducted. The slides were brought into the laboratory and marked off crosswise in narrow strips by running a sharp pencil through the vaseline. These lines served as guides while counting the spores with a microscope.

TABLE I.—*Catches of wind-blown sporangia of Physoderma zeae-maydis at Agricultural College, Miss., in January, 1918*

Stake No.	Date.	Direction of wind.	Relation of stake to cornfield.	Direction slide exposed.	Number of sporangia on slides.		
					1 ft.	2 ft.	3 ft.
1	Jan. 10–13	Storm from northeast, changeable afterwards.	In infested field near diseased plants.	North	2	20	145
1	do	do	do	East	40	9	17
1	do	do	do	South	2	11	7
1	do	do	do	West	12	10	4
2	do	do	At northern edge of infested field.	North	0	2	2
2	do	do	do	East	46	12	17
2	do	do	do	South	57	8	71
2	do	do	do	West	4	9	30
3[a]	Jan. 19–21	Northeast to south.	30 yards from west side of infested field.	North	1	0	0
3	do	do	do	East	2	0	0
3	do	do	do	South	2	0	1
3	do	do	do	West	0	0	0
4	do	do	30 yards from east side of infested field.	North	7	4	1
4	do	do	do	East	3	3	0
4	do	do	do	South	3	2	2
4	do	do	do	West	34	0	0

[a] Stake 3 was blown down during the experiment.

The results given in Table I show that the sporangia of *P. zeae-maydis* may be carried by the wind in considerable numbers. With the abundance of sporangial material which is present in the fields after the corn plants have matured it is quite possible that many of the sporangia are carried by even the very slight breezes.

Further experiments were conducted at Clemson College, S. C., in April, 1918, to obtain additional information on the dissemination of sporangia of the fungus by wind. Slides prepared as mentioned above were placed on stakes without regard to direction in a field where the corn plants had been badly injured by *P. zeae-maydis* in 1917. The plants were cut in the fall of 1917 for shredding, but not until the sporangia had been liberated in large numbers. At the time the slides were placed the field was being planted to cabbage and most of the remaining parts of the corn plants had been plowed under. There was considerable wind and rain during the time the slides were kept in the field. Table II gives the results of these sporangium catches.

TABLE II.—*Results of sporangium catches of Physoderma zeae-maydis on slides placed in fields at Clemson College, S. C., in April, 1918*

Date of experiment.	Stake No.	Number of sporangia per slide at different heights.				
		½ ft.	1 ft.	1½ ft.	2 ft.	2½ ft.
Apr. 4–8	1	8	4	4	21	Lost.
Do	2	8	3	5	6	Lost.
Do	3	18	0	3	3	1
Do	4	Lost.	6	10	Lost.	1

The results given in Table II show that a considerable number of sporangia are carried by wind even after the plants have been removed and the soil has been plowed. These sporangia were doubtless blown from the soil surface as well as from the small parts of diseased plants which remained uncovered. Some of them, however, might have reached the lower slides through spattering rain drops.

Overflowing streams or surface water flowing through infested corn fields after heavy rains may carry large numbers of sporangia to be deposited along their courses. The disease has been found to be more abundant on overflow lands and near streams. Within the infested area the sporangia doubtless reach their host plants in some cases through spattering rain water.

Man is perhaps one of the most important agents by which the fungus is disseminated. In removing diseased plants for stover, fodder, silage, etc., large quantities of sporangia are carried to the barns, and sometimes they may be shipped considerable distances with this material. After these products have been used as feed for animals, the barnyard

manure is utilized as a fertilizer, and no doubt carries with it a large number of viable sporangia which serve as a source of infection to corn plants grown on the land in the future. There is considerable doubt as to the possibility of the sporangia's remaining viable after passing through the silo or the body of the animal, as no experiments have been conducted to determine this point. However, sufficient sporangial material to serve as a means of dissemination escapes the action of the silo and the digestive processes of the stomach of the animal. This would be especially true with stover and fodder, where the tough, thick parts are not eaten by the animals. Hay, unhusked corn, or any other plant products removed from infested fields and shipped to various parts of the country would serve as an ideal means of disseminating *P. zeae-maydis*. This would hold true especially in late fall, when the sporangia are being liberated in so great an abundance.

There is a remote possibility of the dissemination of the fungus to a certain extent by being carried on the seed. However, very little of the disease has been seen, even on the outer husks of the ears. When the ears are husked, the only chance for sporangia to be present on the seed is for them to be brought in by some foreign agent, as they are not produced inside the husks. If the husking is done in the field it is quite probable that some of the loose sporangia will lodge on the husked ears. If, however, a few of them are present on the surface of the kernels of corn when planted the chances are that they will not be able to infect the resulting crop because they are buried with the seed in the drill row, where no cultivation is given and there is practically no chance for them to be disturbed.

If they were to germinate in the soil, the zoospores would have no chance to reach the surface to infect the corn plants. There is a possibility, however, that the sporangia might live over in the soil until the following year, or even longer, and finally come in contact with corn plants, though the possibilities of dissemination in this manner seem to be comparatively small.

POSSIBLE CONTROL MEASURES

No definite means of control has yet been discovered for the disease. However, certain measures may be recommended for reducing the amount of sporangial material present which would have a tendency to reduce the severity of the attacks by the fungus. These measures may be outlined as follows:

(A) The quantity of sporangia present could be greatly reduced by burning the old infected plants after the corn has been harvested, but this would be a destructive practice, especially in the South, where the disease is important and where organic matter is so badly needed in the soil. If the plants could be cut into very fine pieces and plowed under deep enough so that they would not be disturbed by ordinary cultivation the

quantity of infectious material for the following year would be greatly reduced. Where the corn is used for silage or stover, the plants should be cut as near the ground as possible and as early as feasible, in order that a large part of the sporangial material may be removed. Barnyard manure containing these and other corn products should not be used to fertilize corn and should not be put on land where the disease has not been known to occur previously.

(B) Crop rotation may have a tendency to reduce the amount of injury caused by the disease. The most severe cases known have been on land where corn has been grown continuously for a number of years. In a system of rotation the corn plot should be removed as far as possible from the previous plot, as the sporangia of the fungus are wind-borne. Hence, a change of only a short distance in the location of the corn plot probably would have but little effect as a means of reducing the amount of disease present. The longer the duration of the rotation, the better the results are likely to be.

(C) Where the disease continues to be severe, there is a possibility of selecting disease-free plants and from these obtaining a strain that will be resistant to attacks by the fungus. In order to keep them pure, these plants would have to be selected from pure varieties and grown under conditions where crossing could not take place. It seems hardly possible that a variety will be found which naturally resists the disease, as no indications of such a variety have as yet been seen in the various varietal experiments in the South.

(D) Control through seed treatment, no doubt, is worthy of little consideration because there is slight chance for the sporangia to be seed-borne. However, when seed corn is transported from infested territory to territory free from *P. zeae-maydis*, seed treatment would be a desirable precaution, provided an effective method and means of treatment can be found. Sporangia immersed for 10 minutes in a 4 per cent solution of copper sulphate remained viable. This treatment was used to kill the bacteria on the surface of the sporangia.

SUMMARY

(1) The Physoderma disease of corn was discovered in India by Shaw in 1910, and in the State of Illinois by Barrett in 1911.

(2) The disease occurs throughout this country as far westward as central Texas and Nebraska and northward to southern Minnesota and New Jersey. It has perhaps almost reached its northern limits, owing to low temperatures, and its western limits, owing to semiarid conditions.

(3) Considerable damage is caused to corn in the Atlantic and Gulf Coast States and in the Mississippi Valley. The amount of damage varies with weather conditions, moist and warm weather being more favorable for its development.

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(4) The disease is caused by a species of Physoderma, one of the Phycomycetes, which is probably identical with Shaw's Indian species, *P. zeae-maydis*.

(5) The sporangia of the fungus live over the winter on the old, diseased corn plants and in the soil, and germinate the following summer by producing numerous zoospores which infect the corn plants and cause the disease.

(6) The sporangia require free water and a high temperature, 23° to 30° C., for germination and host penetration.

(7) The fungus is disseminated by wind and probably by other agencies—for example, flowing water, insects, and various animals, including man.

(8) There is a possibility of controlling the disease to a certain extent through sanitation, crop rotation, and resistant varieties, although this has not been positively proved.

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PLATE A

Corn leaf showing the effects of an attack by *Physoderma zeae-maydis*. Notice the light-brown, rustlike appearance on the thin parts of the blade and the larger, dark spots on the midrib. This difference in color is due to the fact that a greater number of the dark-brown sporangia are produced in the fleshy tissues of the midrib.

PLATE B

Sheath and culm of corn plant showing the effects of *Physoderma zeae-maydis*. In these parts there is a large production of sporangia, which causes the dark spots, as is true in case of the midrib of the blade as seen in Plate A. In some cases the entire sheath is darkened and the nodes completely girdled by the fungus.

PLATE 10

Blade and sheath of corn plant showing the effects of a severe attack by *Physoderma zeæ-maydis*.

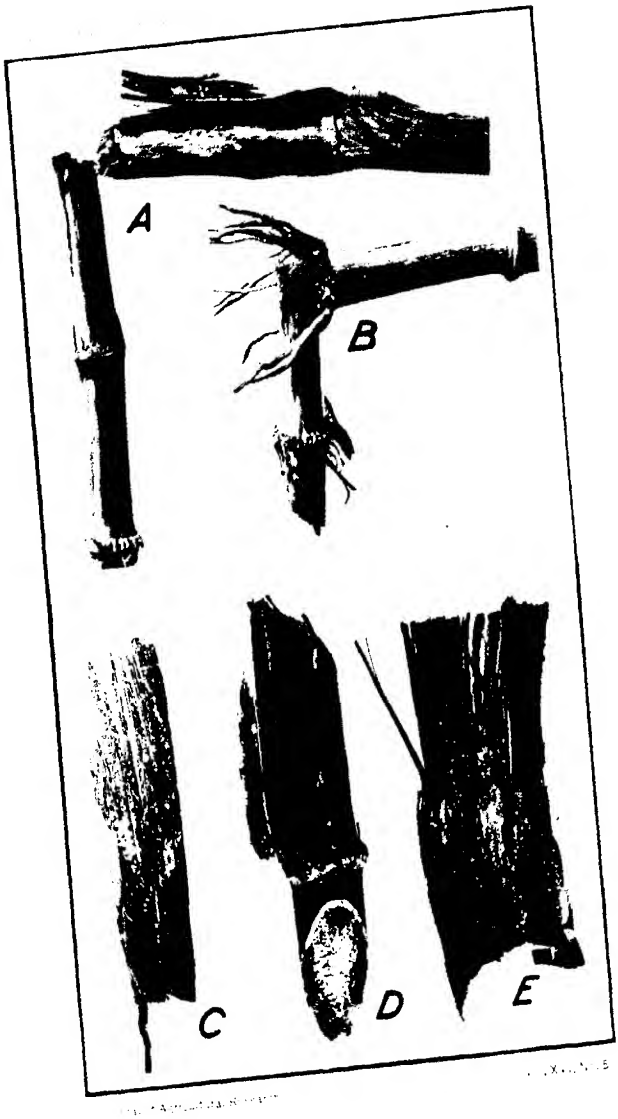

A
B
C
D
E

PLATE 11

Old sheaths and culms of corn showing effects of severe attacks by *Physoderma zeæ-maydis*:

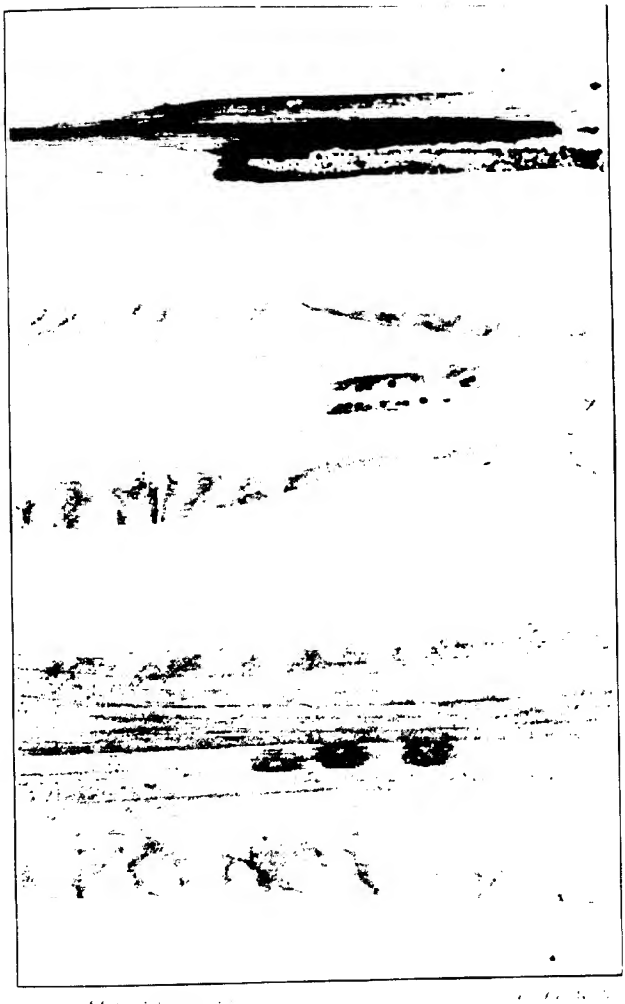
A, B.—Badly diseased stalks broken over at weakened, infected lower nodes. These are the effects of the disease in regions where it is most destructive.

C, E.—Portions of old attacked sheaths showing characteristic shredding. These old infected sheaths contain countless numbers of resting spores which become freed and are then blown and washed about when sheaths become weathered and shredded.

D.—Portion of an old infected stalk showing discoloration both on outside and in pith due to the attacks of the fungus. Numerous spores are found in all attacked portions.

PLATE 12

Blades and sheath of corn showing the Physoderma disease produced by inoculating plants in the greenhouse with a suspension of the sporangia of *P. zeae-maydis* in tap water. The suspension was poured behind the sheaths and in the bud of the plants and the plants were sprayed twice daily to keep them damp. Notice the dark area around the diseased tissue in the bottom figure. This is a dark-red color which is often seen on diseased plants in the field.




a
b
c
d
f
g
h

PLATE 13

Physoderma zeae-maydis: Various stages in the germination of sporangia, formation of zoospore, and germination of zoospore.

a.—Sporangium.

b, *c*, *d*.—Opening sporangia showing the early stages of zoospore formation.

e.—Mature zoospores escaping through the ruptured apex of the endosporangium.

f.—The collapsing endosporongium after the zoospores have escaped. A single zoospore remained within the sporangium. The active stage of zoospores remaining within the sporangium was easily studied.

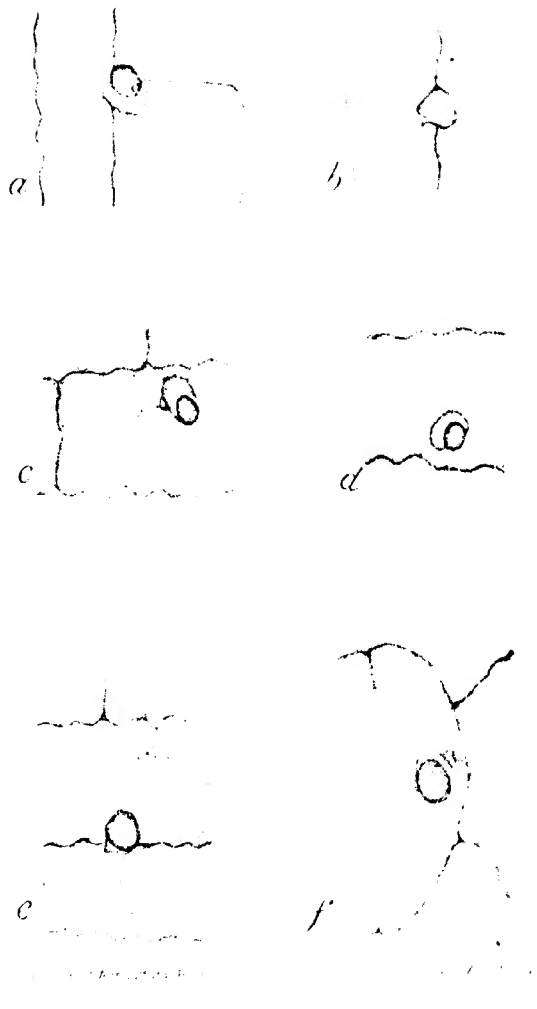
g.—Zoospores.

h.—Germinating zoospores.

PLATE 14

Physoderma zeæmaydis:

a–f,—Zoospores germinating by fine threadlike hyphæ which have penetrated the epidermal cell walls of a tender leaf of Indian corn. In *c*, *e*, and *f* the enlarged cells have begun to form in the epidermal cells of the host.


a
b
c
d
e
f


a
b
c
d
e
f

PLATE 15

Physoderma zeae-maydis: Mycelial stages within the host cells.

a–d.—Drawings from ordinary high-power magnifications showing the fibers and enlarged cells of the mycelium.

e–g.—Drawings magnified with oil-immersion lens.

b, d, g.—Notice the young sporangia at the ends of the short hyphæ.

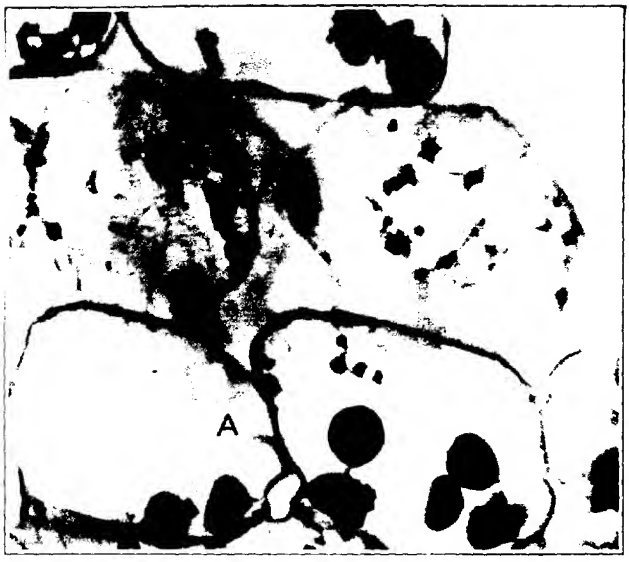
PLATE 16

Physoderma zeae-maydis:

a–e, Mycelial fibers penetrating the cell walls of the host tissue.

f, *g*, Different types of reproductive bodies. Notice the double nucleate condition in figure *g*.


a
b
c
d
e
f
g


A

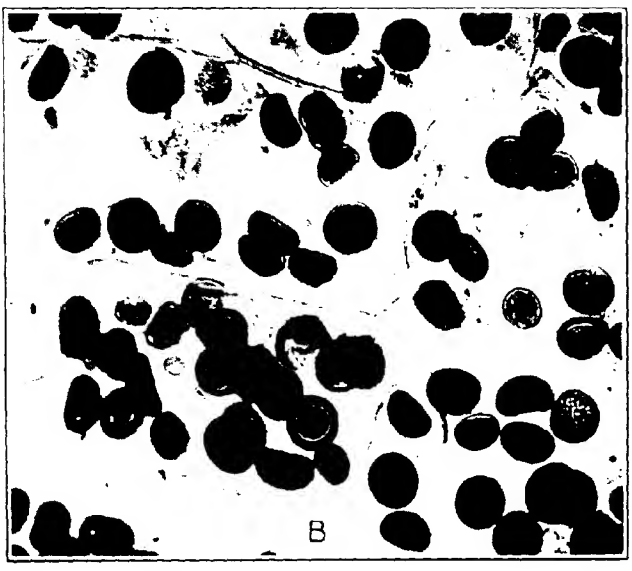

B

PLATE 17

Physoderma zeae-maydis: Photomicrographs showing the different stages of the development in the host tissue (teosinte).

A.—Notice the reproductive bodies connected by the very fine threadlike hyhae in the central cells of the figure.

B.—Host cells filled with mature sporangia.

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